# Endovascular Transvenous Embolization Combined with Direct Punction of the Sinus Pericranii

# A Case Report

I.M. KESSLER, B. ESMANHOTO, R. RIVA, C. MOUNAYER

Neurointerventional Unit, Dupuytren University Hospital; Limoges, France

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# **Summary**

Sinus Pericranii is a rare venous anomaly in which the communication between the intraand extracranial venous system is constituted by a network of thin-walled veins that form a varix on the external table of the skull. This varix is continuous with the pericranial veins of the scalp. We describe a 31-year-old woman with frontal sinus pericranii treated using an original endovascular technique by transvenous approach combined with direct punction.

#### Introduction

Sinus pericranii (SP) was first published by Percival Potts in 1760<sup>1</sup>, but the term sinus pericranii was introduced in 1850 by Stromeyer<sup>2</sup> who described it as a "blood bag on the skull in connection with the veins of the dipole and through these with the sinuses of the brain".

The etiology of SP is unknown. However, its frequent association with intracranial developmental venous anomalies (DVA) or other anomalies has led authors to support a congenital cause<sup>3</sup>. Treatment of SPs by open surgery or endovascular approaches has been generally recommended for cosmetic reasons and prevention of hemorrhage<sup>4</sup>.

We describe a successful case of SP treated by an original endovascular technique combining transvenous approach and direct puncture.

# **Case Report**

A 31-year-old woman was admitted with a cosmetic complaint of a 5x5 cm right frontal, superficial, non-pulsatile, painless soft tissue mass. It had been increasing slowly during the last 12 years. There was no history of trauma and the skin over the lesion was normal as was neurologic examination. The mass was observed to increase in size during a Valsalva maneuver.

MRI (Figure 1A) and CT scan with 3D reconstruction (Figure 1B) showed a vascular feature of the lesion.

The procedure was performed under general anesthesia. An IV injection of 3000 IU of heparin was infused at the beginning of the treatment. After a right femoral artery 5 French sheath placement, a full angiogram including both internal carotid arteries (ICAs), external carotid arteries (ECAs) and vertebral arteries selective catheterization was performed (Figure 2A). A superseletive venogram was obtained from direct punction of the varix using 21 Gauge needles (Figure 2B). It showed the extra skull venous pouch as well the emissary vein connecting the SP with the superior sagittal sinus (SSS).

A right jugular vein 5 French sheath was placed and a 5 French guiding catheter was navigated near the torcular sinus. Under a road map acquisition from the direct punction of the

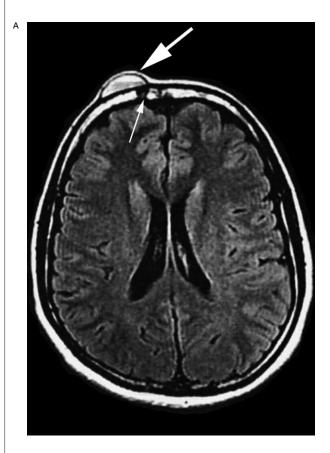




Figure 1 A) Flair T1 weighted MRI shows a mass in the scalp (thick arrow) overlying a small defect in the right frontal bone (thin arrow). Its hypersignal testifies that the lesion flow is very slow. B) Reconstructed 3D CT images shows the mass overlying the right frontal bone and demonstrate curvilinear grooves within the skull, corresponding to the course of the large draining veins.

SP, an Echelon 10 microcatheter (eV3 Neurovascular, Inc.) was coaxially navigated through the guiding catheter and its distal tip was placed distally in the emissary vein connecting both the SP and SSS (Figure 3A).

To obtain a total disconnection between the varix and the SSS, we deployed through the microcatheter two Guglielmi detachable coils (GDC® coils) 10; (Boston Scientific, Fremont CA, USA) (3 mm x 8 cm) and (2 mm x 8 cm) followed by 0.5 ml of acrylic glue injection (Glubran -2, GEM, Viareggio, Italy) mixed 4:1 with lipiodol (Figure 3B). An ICA angiogram was performed showing the absence of coil or glue protrusion inside the SSS. The venogram showed the total occlusion of the diploe vein, disconnecting the varix of the SSS (Figure 4). After these observations, 5 cc of absolute alcohol were directly injected in the varix. The postoperative course was uneventful and the patient was discharged three days after the procedure and had had no signs of recurrence six months later (Figure 5).

#### **Discussion**

## Clinical Presentation

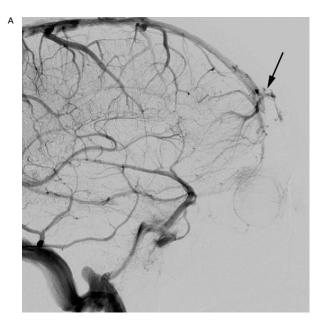
Sinus pericranii usually first appears in child-hood and young adulthood, especially if it is congenital in origin<sup>5</sup>. The incidence is equal between men and women, except in cases attributed to trauma in which males outnumber females 2:1<sup>6</sup>.

The SSS is the most commonly involved of the dural sinus: hence most SP lesions tend to be located near the midline of the skull. Lateral SP lesions are rarer but have been reported in the literature <sup>7</sup>. The defect most commonly lies in the frontal bone (40%), the parietal bones (34%), the occipital bones (23%) and rarely, the temporal bones (4%)<sup>8</sup>.

SP typically presents as a slowly enlarging scalp mass, located near the midline, which is soft non pulsatile and fluctuant. The mass enlarges with activities that increase intracranial pressure. The mass decreases in size or disappears when the patient stands 9.

#### Diagnosis

Sinus pericranii should be suspected in a patient who is first seen with a fluctuant subgaleal mass overlying the skull that enlarges with increased intracranial pressure, as seen in Valsava



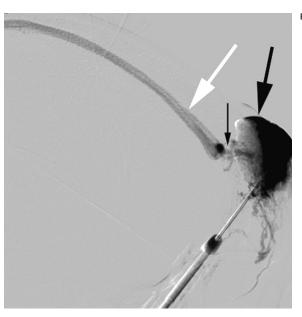


Figure 2 A) Right ICA selective angiogram (lateral view) during its venous phase shows a connection between a diploic vein and the SSS (arrow) although the entire venous pouch could not be identified. The recumbent position and the presence of a venous valve could explain these findings. B) The venogram (lateral view) shows the anterograde filling of the pouch by the contrast medium (thick black arrow), the diploe vein (thin black arrow) and the SSS (white arrow).

maneuvers and placement of the head in a dependent position. The mass disappears with standing and is fully reducible with compression. Palpation of the skull underlying the mass may reveal a bone defect, and aspiration of the mass often reveals venous blood.

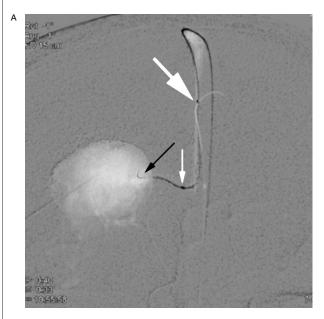




Figure 3 A) Road map acquisition (AP view) from direct contrast medium injection through the varix shows an Echelon 10 microcatheter (eV3 Neurovascular, Inc.) retrogradely navigated via the SSS and a selective diploe vein catheterization.. Note the tip of the guidewire (thin black arrow) in the pouch, the distal (white thin arrow) and the proximal (white thick arrow) markers of the microcatheter. B) Road map acquisition (AP view) from direct contrast medium injection through the varix shows coils deployment (arrow) and n-BCA injection in the diploe vein.

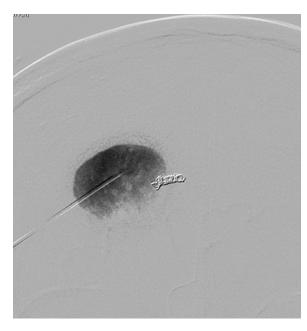


Figure 4 Venogram (AP view) shows 5 ml injection of pure alcohol in the SP after the total disconnection of the varix from the SSS.

Diagnosis is usually confirmed radiographically. CT scan generally reveals not only a subcutaneous mass which is enhanced by intravenous contrast, but also the degree and extent of bone erosion on the modality of bone win-

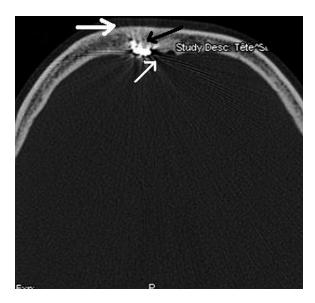


Figure 5 CT scan, bone window, performed six months after the procedure. Note the metal density of the coils (thin white arrow), the total reossification of the skull (black arrow) and the disappearance of the pouch under the skin (thick white arrow).

dows. MRI reveals a lesion of heterogeneous signal intensity, with flow void signal that represents blood flow; it also may show the relation of a dural sinus subjacent to the lesion <sup>10</sup>.

DSA shows connections between the lesion and the involved dural sinus <sup>11</sup>, and helps distinguish the more serious type of SP (i.e., herniated dural sinus, which has the potential for hemorrhage) from the less precarious types (vascular hematomas and cutaneous venous varix), it is the best method to exclude other vascular entities and for strategy of treatment <sup>11</sup>.

The differential diagnosis is extensive because several congenital and posttraumatic scalp lesions can mimic SP. Vascular lesions (subepicranial varix, venous cavernoma, and arteriovenous fistula), traumatic leptomeningeal cysts, subepicranial hygroma, cephaloceles (meningocele, meningoencephalocele, and myelomeningocele), dermoid tumors, eosinophilic granulomata, meningeoma, neoplasm, fibrous dysplasia, skull bone hemangiomas are the diseases that can mimic SP <sup>12</sup>.

### Treatment

Treatment of SP is usually for cosmetic reasons, but prophylactic correction of the defect by a neurosurgeon is advised to prevent complications such as hemorrhage, infection, or traumatic air embolism <sup>13</sup>.

Gandolfo et Al14 classified SP based on flow dynamics identified at angiography into two basic patterns: 1) "dominant" in which the main stream of contrast flow uses the SP to drain the brain parenchyma, and 2) "accessory" in which only a portion of the brain's venous outflow occurs through the extradiploic vessels. A dominant flow pattern is considered a contraindication to both surgical and endovascular treatment, in view of the potential complications, including bleeding, venous congestion and/or infarction, and hemorrhage. The feasibility of treatment of SP with an accessory flow pattern is dependent on the degree of brain parenchymal venous drainage into the dural sinuses, as demonstrated on angiography. Normal brain parenchymal venous drainage cannot be compromised.

The gold standard method of treatment of SP is defined as the surgical procedure, but there are studies series that relate laceration of the dural sinus with consequent hemorrhage as a complication of this treatment modality <sup>15</sup>. It involves removal of the extracranial mass and

closure of the emissary veins by means of bone wax. Radical operations involving craniotomy or craniotomy and cranioplasty to correct the skull defect have also been described <sup>16</sup>.

According to Nakasu et Al17, findings on histologic examination and imaging studies will separate SP into three different types: 1) vascular hematomas, multilocular blood cyst consisting of fibrous connective tissue similar to the architecture of cavernous hemangioma; 2) cutaneous venous varix, and 3) herniated dural sinus through the defect of calvarium. These distinctions are important in determining management. Herniated dural sinus may be suspected when the bony defect is large, and care must be taken to prevent massive hemorrhage during repair of this lesion. Regarding a venous varix, histological examination reveals a venous lesion mimicking sinus pericranii and containing endothelial cells but no communication with the intracranial venous sinuses is identified 18.

Endovascular surgery for SP is another means of treatment that, despite being less invasive than conventional surgery, carries the risk of causing necrosis of the overlying skin or embolic events <sup>19</sup>. Some authors like Brook et Al<sup>20</sup> report a definitive occlusion of SP treated by an endovascular transvenous route using *n*-BCA as embolic agent.

The effect caused by *n*-BCA is a simple vascular occlusion because this agent cannot entirely destroy the endothelial cells of the vessel and is not reabsorbed. This event induces an inflammatory chain and cellular infiltration into the vascular channels. Once this mechanism occurs, the endothelial cell is reconstituted and

recanalization can occur  $^{21}$ . Another issue is that injection of n-BCA into the entire SP may result in undesirable cosmetics results because it remains as a non absorbed embolic material.

In our case, a transvenous route was performed and the lesion was embolized combining coils and n-BCA followed by direct punction of the SP and injection of pure alcohol. This embolic agent denudes the endothelial cell from the vascular wall, and there is a rupture in the vascular wall to the level of the internal elastic lamina. These changes are desirable and account for the curative and permanent effects of ethanol embolization. Despite reports of potential problems with ethanol embolization, we are confident about absolute ethanol use in the management of SP through direct puncture embolization. The complications can be avoided considering controlled delivery and proper dosage .The final cosmetic result is usually excellent and definitive.

#### **Conclusions**

Sinus pericranii is a rare congenital or acquired disorder characterized by extracranial vascular lesions with anastomotic connections to an intracranial dural sinus.

Although open surgery management of SPs is generally recommended as a gold standard, it can be related to hazardous hemorrhagic complications.

Endovascular treatment of SPs combining transvenous approach and direct puncture embolization is a less invasive, feasible, safe and highly effective method.

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Dr Charbel Mounayer Department of Neurointerventional Unit Dupuytren University hospital Limoges, France E-mail: neuroradiologie@chu-limoges.fr